monoterpene toxic to TA100 strain in the preliminary toxicity test. No mutagenic effect was found with (±) camphor, citral, citronellal, 1,8-cineole, and (−)-menthol. Terpineol caused a slight but dose-related increase in the number of his+ revertants with TA102 tester strain both without and with addition of S9 mixture. The results from this study therefore suggest that, with the exception of terpineol, the monoterpenoid compounds tested are not mutagenic in the Ames test''. Thus not all the essential oil compounds are mutagenic.

The antifungal activity of the essential oils of *Thymus* on *Candida spp* has been described as warranting future therapeutic trials on mucocutaneous candidosis (Pina-Vaz et al.[1]). Paster and co-workers[2] applied essential oils from thyme for 24 hours against the mycelia and spores of *Aspergillus flavus*, *Aspergillus niger*, and *Aspergillus ochraceus*, as well as against the natural microflora of wheat grains. The thyme essential oil was less efficient in controlling mycelia and growth was observed even following exposure to 4.0 mU/l. However, the thyme essential oil was fungitoxic to spores (MIC = 3.0 mU/l). In another set of trials the efficacy of the oils and two of their constituents (carvacrol and thymol) in controlling the natural microflora of surface-sterilized wheat grain was studied. Juliano et al.[3] demonstrated that thyme oils possessed microbicidal activities, especially against Gram-positive bacteria (MIC range 0.125—0.500 mg/ml) and fungi (MIC 0.125—0.500 mg/ml). Soliman and Badeaa[4] reported complete inhibition of *Aspergillus flavus*, *Aspergillus parasiticus*, and *Aspergillus ochraceus* by the oils of thyme and cinnamon (<500 ppm), marigold (<2000 ppm), spearmint, basil, and quyssum (3000 ppm). There is a strong correlation between the contents of camphor, thujones and camphene, and the oils' toxicity. The spring extract was the least toxic (LD_{50}: 1200 mg/kg body weight) and contained lower levels of camphor (7.7%), α,β-thujone (1.3%), and camphene (3.1%). Thus, we recommend that oil extracts of sage marketed for use in certain unconventional medicines be prepared from spring plants (Farhat et al.[5]). Owing to strong antibacterial and excellent protective features exhibited in antioxidant activity tests, the essential oil and extracts from the herbal parts of *Thymus eigii* were suggested for consideration as a natural source that can be freely used in the food industry as a culinary herb (Tepe et al.[6]).

Conflict of interest: No conflict of interest to declare.

References

1. Pina-Vaz C, Gonçalves Rodrigues A, Pinto E, Costa-de-Oliveira S, Tavares C, Salgueiro L, et al. Antifungal activity of *Thymus* oils and their major compounds. *J Eur Acad Dermatol Venereol* 2004;**18**: 73—8.
2. Paster N, Menasherov M, Ravid U, Juven B. Antifungal activity of oregano and thyme essential oils applied as fumigants against fungi attacking stored grain. *J Food Prot* 1995;**58**:81—5.
3. Juliano C, Mattana A, Usai M. Composition and in vitro antimicrobial activity of the essential oil of *Thymus herba-barona* Loisel growing wild in Sardinia. *J Essential Oil Res* 2000;**12**:516—22.
4. Soliman KM, Badeaa RI. Effect of oil extracted from some medicinal plants on different mycotoxigenic fungi. *Food Chem Toxicol* 2002;**40**:1669—75.
5. Farhat GN, Affara NI, Gali-Muhtasib HU. Seasonal changes in the composition of the essential oil extract of East Mediterranean sage (*Salvia libanotica*) and its toxicity in mice. *Toxicon* 2001;**39**:1601—5.
6. Tepe B, Daferera D, Sokmen M, Polissiou M, Sokmen A. In vitro antimicrobial and antioxidant activities of the essential oils and various extracts of *Thymus eigii* M. Zohary et P.H. Davis. *J Agric Food Chem* 2004;**52**:1132—7.

Iraj Rasooli*
Department of Biology, Shahed University, PO Box 18151-159, Tehran, Iran

*Tel.: +98 21 55277509; fax: +98 21 55277645
E-mail address: rasooli@shahed.ac.ir

Corresponding Editor: Jonathan Cohen, Brighton, UK

11 August 2006

doi:10.1016/j.ijid.2006.08.004

Cryptococcal meningitis with raised intracranial pressure masquerading as malignant hypertension

A 39-year old man had recently been diagnosed with hypertension and was taking nifedipine SR. He presented with neck pain and diffuse headache of one week's duration, and had blurred vision for two days; he was afebrile. His blood pressure (BP) was 189/118 mmHg and retinal flame-shaped hemorrhages were seen without other abnormalities. An electrocardiogram and plain computerized tomography brain scan were normal. His blood pressure was controlled and the headache improved with hypotensive agents. Routine blood tests and screening for causes of secondary hypertension were normal or negative.

Three weeks later he developed confusion and diplopia. Examination revealed nuchal rigidity with Glasgow coma scale 13 (M5E4V4), impaired abduction of both eyes, sluggish left pupil, papilloedema, and flame-shaped hemorrhages. His BP was 199/118 mmHg. A magnetic resonance imaging brain scan with gadolinium was normal. Lumbar puncture showed an opening pressure of 52 cmH_2O. Cerebrospinal fluid (CSF) protein was 2.3 g/l, glucose was 2.2 mmol/l (blood 6.8), and cell count was 87×10^6/l (88% lymphocytes); the CSF was positive for *Cryptococcus neoformans* on microscopy with elevated cryptococcal antigen (1:32). Amphotericin and flucytosine were started and ventriculostomy performed for continuous intracranial pressure (ICP) monitoring. The lowering of his BP to 138/90 mmHg with nifedipine SR was followed by deteriorated consciousness and confusion, which improved when his BP surged up again. Transcranial Doppler performed during normal BP revealed reduced flow velocities over the major intracranial arteries, which rose to normal when his BP surged up to 185/103 mmHg. His mental state, ICP, and BP

improved subsequently with full recovery. Amphotericin and flucytosine were stopped after two weeks and were followed by oral fluconazole for six months. He was HIV negative. Lymphocyte subsets count and lymphocyte proliferation assay were normal. He had no relapse for five years.

Cryptococcosis is an important diagnosis for immunocompromised subjects,[1,2] and is increasingly recognized in the immunocompetent host.[3,4] Meningitis is common in cryptococcosis.[2,5–8] Our patient had cryptococcal meningitis presenting with neck pain and headache with raised ICP. The raised ICP aggravated his hypertension via the Cushing reflex. During the Cushing reflex, the systemic arterial blood pressure rises in response to central nervous system (CNS) ischemia caused by an ICP that is equal or higher than the arterial pressure. Without end-organ damage, we believed that his papilloedema was due to raised ICP instead of malignant hypertension. CNS infection was not considered initially due to the lack of fever and the presumptive diagnosis of headache due to uncontrolled hypertension. The clue for CNS infection on initial presentation was the neck pain. He deteriorated later with encephalopathy, nuchal rigidity, and severe intracranial hypertension causing papilloedema and sixth nerves compression. Upon incidental normalization of his BP, his level of consciousness deteriorated, which improved as his BP surged up, reflecting that when cerebral autoregulation is impaired due to meningitis, a high systemic BP is required to maintain adequate cerebral blood flow during intracranial hypertension. This was documented objectively by the transcranial Doppler findings.

Raised ICP causing reactive hypertension without fever or signs of meningism can be the presenting feature of cryptococcal meningitis in an immunocompetent host. Clinicians should consider CNS infection causing raised ICP when hypertension and papilloedema are present without evidence of end-organ damage to suggest malignant hypertension. Prompt initiation of anti-fungal therapies is critical for this potentially fatal meningitis.

Conflict of interest: No conflict of interest to declare.

References

1. Diamond RD, Bennett J. Prognostic factors in cryptococcal meningitis. *Ann Intern Med* 1974;**80**:176–81.
2. Lu CH, Chang WN, Chang HW, Chuang YC. The prognostic factors of cryptococcal meningitis in HIV-negative patients. *J Hosp Infect* 1999;**42**:313–20.
3. Lui G, Lee N, Ip M, Choi KW, Tso YK, Lam E, et al. Cryptococcosis in apparently immunocompetent patients. *QJM* 2006;**99**:143–51.
4. Kiertiburanakul S, Wirojtananugoon S, Pracharktam R, Sungkanuparph S. Cryptococcosis in human immunodeficiency virus-negative patients. *Int J Infect Dis* 2006;**10**:72–8.
5. Gottfredsson M, Perfect JR. Fungal meningitis. *Semin Neurol* 2000;**20**:307–22.
6. Yu YL, Lau YN, Woo E, Wong KL, Tse B. Cryptococcal infection of the nervous system. *QJM* 1988;**66**:87–96.
7. Jean SS, Fang CT, Shau WY, Chen YC, Chang SC, Hsueh PR, et al. Crytococcaemia: clinical features and prognostic factors. *QJM* 2002;**95**:511–8.
8. Saag MS, Graybill RJ, Larsen RA, Pappas PG, Perfect JR, Powderly WG, et al. Practice guidelines for the management of cryptococcal disease. Infectious Diseases Society of America. *Clin Infect Dis* 2000;**30**:710–8.

K.H. Chan*
W. Mak
S.L. Ho
University Department of Medicine,
4/F Professorial Block,
Queen Mary Hospital,
The University of Hong Kong,
Pokfulam Road, Hong Kong
*Corresponding author. Tel.: +852 2855 5318; fax: +852 2855 5322
E-mail address: koonhochan@hotmail.com
(K.H. Chan)

Corresponding Editor: Andy I.M. Hoepelman, Utrecht, The Netherlands

5 May 2006

doi:10.1016/j.ijid.2006.07.005

The use of ELISA in a seroprevalence study of *Brucella* antibodies in West Bank Palestinian women of childbearing age

Brucellosis is considered to be endemic in the Mediterranean basin and the Middle East;[1,2] *Brucella melitensis* is the only *Brucella* species currently known to cause disease in Israel and the Palestinian Authority.[3] While human incidence statistics from Israel and the Palestinian Authority support endemicity,[4,5] this has not previously been verified by seroprevalence studies. We report a prevalence study of *Brucella* antibodies in a West Bank Palestinian population.

Cord blood was taken at delivery, after informed consent, from 2638 Palestinian women who gave birth between 31 August 1994 and 31 March 1995 in six West Bank government hospitals. Of 1021 samples selected by systemic sampling, 802 were adequate for processing. All were tested by ELISA for IgG-class *Brucella* antibodies (Clark). Every sixth sample, and all ELISA-positive samples with sufficient serum (32/35 positive samples), were tested by Rose-Bengal (Sanofi) and agglutination (SAT; Murex) tests.

Of the samples, 35/802 (4.4%) were positive by ELISA. Nine of 32 (28.1%) ELISA-positive samples had SAT titers $\geq$1:50; one (1:400 titer) was positive by Rose-Bengal test. All ELISA-negative samples were SAT-negative. ELISA prevalence varied from 3% in the summer to 7–10% in early spring (Figure 1). Neither this nor variation by age (from 2.5% for ages 20–24 years to 7% in the $>$30 years age group) or hospital (1.3–6%) was statistically significant.

ELISA detects *Brucella* IgG antibodies reflecting both past seroconversion and current disease.[2,6,7] ELISA tests combine high specificity and sensitivity and a relatively low unit